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PULMONARY ASPIRATION WAS DETECTED BY DECREASE IN TRANSCUTANEOUS OXYGEN TENSION DURING SEDATION: A CASE REPORT

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ABSTRACT

Background: We report a case with pulmonary aspiration during surgery under epidural and spinal anesthesia with sedation, whose pulmonary aspiration was suspected by decrease in transcutaneous oxygen tension ($tcPO_2$). **Case Report:** A 70 years old man received axillo-femoral bypass grafting and thrombendarterectomy of femoral artery under epidural and spinal anesthesia. $TcPO_2$ and transcutaneous carbon dioxide tension ($tcPCO_2$) were monitored at his right chest. Percutaneous oxygen saturation (sPO_2) was monitored at the right index finger. He was sedated with midazolam and propofol. Two hours after start of anesthesia, $tcPO_2$ suddenly decreased without remarkable change in SpO_2. Arterial oxygen pressure (PaO_2) decreased to 73 mmHg from 345 mmHg. Chest computed tomography (CT) after surgery showed consolidation mainly in the right lung, which suggested pulmonary aspiration. **Conclusions:** $TcPO_2$ was better than SpO_2 to detect decrease in PaO_2 by pulmonary aspiration during surgery under epidural and spinal anesthesia with sedation.

KEYWORDS: Aspiration pneumonia, transcutaneous oxygen tension, percutaneous oxygen saturation.

INTRODUCTION

The systematic review of pulmonary aspiration during procedural sedation showed few occurrences with full recovery except for gastrointestinal endoscopy.[1] Decrease in arterial oxygen pressure (PaO_2) is important to suspect pulmonary aspiration during sedation. However, it is usually difficult to monitor PaO_2 continuously during sedation. Therefore, percutaneous oxygen saturation (SpO_2) is ordinally used. The transcutaneous oxygen tension ($tcPO_2$) monitoring is a non-invasive continuous assessment of arterial oxygenation. Heating skin causes local vasodilation and increases cutaneous capillary blood flow. Then, the oxygen tension at the skin increases to a level similar to PaO_2.[2] We have already shown that $tcPO_2$ measured with the electrode on the chest well correlated well with PaO_2 during general anesthesia.[3] We report a case with pulmonary aspiration during surgery under epidural and spinal anesthesia with sedation, whose pulmonary aspiration was detected by decrease in $tcPO_2$.

CASE REPORT

A 70 years old man with weight 63 kg and height 162 cm was scheduled for axillo-femoral (AP) bypass grafting and thrombendarterectomy of femoral artery for stenosis of anastomosis of femoro-popliteal (FP) bypass. He had intermittent claudication of his left leg and received FP bypass for occlusion of bilateral superficial femoral artery 5 years ago. Three years and 3 months later, he had intermittent claudication again, and stenosis of left anastomosis of FP bypass was observed. One year later, he received coronary arterial bypass grafting for unstable angina pectoris and remove and regrafting of stenosis of FP bypass. Nine months later, he was scheduled for re-AP bypass and thrombendarterectomy of femoral artery. He had diabetes mellitus using insulin, bronchial asthma, angina pectoris, and hyperlipidemia. Preoperative examination showed HbA1C 7.8%, forced expiratory volume (FEV)1.0% 63% and asynergy in middle left ventricle and posterior wall in echocardiogram. No other abnormality was found.

He received no premedication. An epidural catheter was inserted at T12/L1 intervertebral space with median approach in the right lateral position. A catheter was inserted 5cm ahead. Then spinal anesthesia was done at L4/5 intervertebral space with median approach using hyperbaric tetracaine 13 mg. Anesthesia level was Th8 by the cold test. Transcutaneous oxygen ($tcPO_2$) and carbon dioxide ($tcPCO_2$) were monitored at his right

chest using TCM4^{TM} (Radiometer, Copenhagen, Denmark). Percutaneous oxygen saturation (sPO_2) was monitored at the right index finger. An arterial catheter was inserted into the left radial artery to measure blood pressure and analyze blood gas. Under oxygen 6 L/min by a face mask, he was sedated with midazolam 1mg and propofol 2 mg/kg/h. An epidural infusion was started at 0.2% ropivacaine 5 mL/h with fentanyl 15 µg/h. Two hours after start of anesthesia, $tcPO_2$ suddenly decreased without remarkable change in SpO_2 (Fig.1). Arterial oxygen pressure (PaO_2) decreased to 73 mmHg from 345 mmHg. Then, propofol was stopped. Chest computed tomography (CT) after surgery showed consolidation mainly in the right lung (Fig.2). He was admitted into the high care unit. Hydrocortisone, cefazolin and clindamycin were administered. Twenty hours after surgery, cricothyrotomy was performed to suction sputum and it was removed 20 hours later. He was discharged from high care unit to the ward 42 hours after surgery (Fig.3).

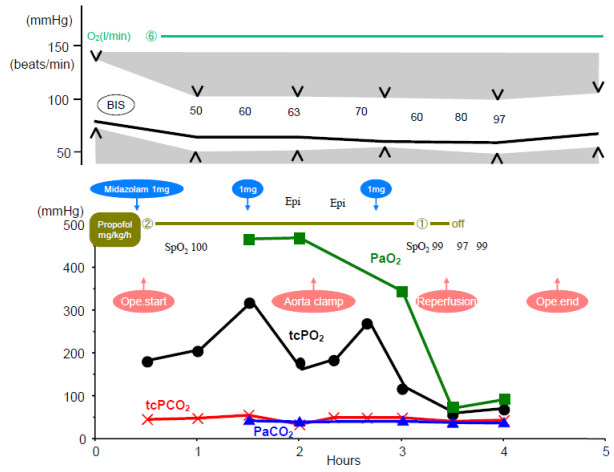

Fig. 1. Anesthesia record BIS, Bispectral index; SpO_2, percutaneous oxygen saturation; PaO_2, arterial oxygen pressure; $PaCO_2$, arterial carbon dioxide pressure; $tcPO_2$, transcutaneous oxygen tension; $tcPCO_2$, transcutaneous carbon dioxide tension.

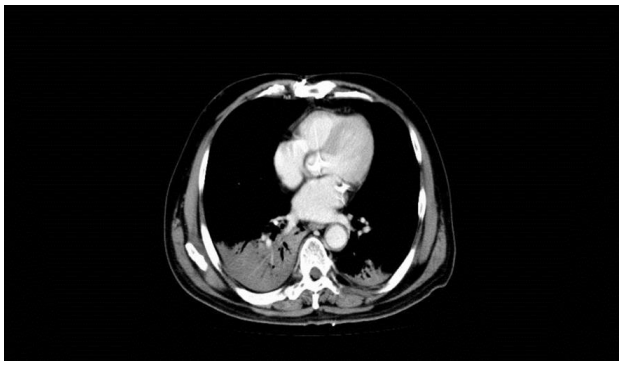

Fig. 2. Chest computed tomography after surgery Air space consolidation was observed, especially in the right lung.

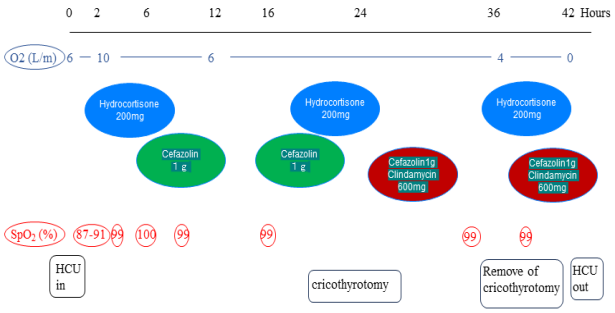

Fig. 3. Postoperative course in the high care unit HCU, high care unit.

DISCUSSION

In this case, we measured intermittent PaO_2 and $PaCO_2$, and continuous SpO_2, $tcPO_2$, and $tcPCO_2$. In these monitors, $tcPO_2$ decreased first, and SpO_2 and $tcPCO_2$ did not change significantly. Then we confirmed decreased PaO_2.

An animal study showed that a minimum of 0.8 mL/kg of gastric acid with a pH < 2.5 was required to produce lung changes consistent with aspiration.[4] Our case was sedated and there were no cough and movement and SpO_2 did not change when $tcPO_2$ decreased, therefore, we might not be able to detect aspiration during surgery if we did not monitor $tcPO_2$.

$TcPO_2$ was reported to correlate well with PaO_2 during anesthesia.[5] The regression slope of 0.85 and correlation coefficient 0.92 between $tcPO_2$ and PaO_2.[6] In a case report, during cardiac arrest and resuscitation, once the patient was resuscitated $tcPO_2$ closely followed PaO_2.[7] However, Stosseck et al. showed that $tcPO_2$ tend to be lower than PaO_2 about 15%[8] because of oxygen consumption by the skin.[9] In our case, difference between $tcPO_2$ and PaO_2 were large when PaO_2 were higher than 300 mmHg, but both were very close when PaO_2 were less than 100 mmHg. This is consistent with the report by Hutchison et al. that in the patients with chronic respiratory disease, measurement of $tcPO_2$ can estimate PaO_2 in the range 50 to 100 mmHg.[9]

During hypotensive anesthesia, supraclavicular $tcPO_2$ could give accurate values, while forearm $tcPO_2$ was the least sensitive.[10] This case did not have hypotension, and we put the electrode of $tcPO_2$ on the chest because we previously showed the chest was better to correlate with PaO_2 than the arm.[3] There was a significant correlation between change in mean arterial pressure and change in $tcPO_2$ after ephedrine administration.[11] In this case, we did not administer any vasoactives and blood pressure did not change significantly, therefore, decrease in $tcPO_2$ was not due to changes in metabolism and hemodynamics.

During stable respiration, $tcPO_2$ correlated well with PaO_2. However, when PaO_2 changes rapidly $tcPO_2$ could

not correlate with PaO_2. A lag time was more than 5 minutes for $tcPO_2$ to follow sudden changes in oxygenation.[12] However, change in $tcPO_2$ occurred more rapidly than that in SpO_2; median times for change in $tcPO_2$ of 120 seconds and SpO_2 of 174 seconds,[13] while Van Wetringen et al. showed that the response time of $tcPO_2$ was about 2 minutes longer than SpO_2.[14] The changes in SpO_2 were smaller than those in $tcPO_2$ in our case. This is the same as in patients undergoing bronchoscopy who are receiving oxygen.[13] Thus, $tcPO_2$ is better than SpO_2 to detect sudden change of PaO_2.

Continuous monitoring makes $tcPO_2$ possible to detect acute changes, but not $tcPCO_2$.[15] During early phase of hypoventilation or hyperventilation, $PaCO_2$ and $tcPCO_2$ will not correlate well.[12] During air embolism, $tcPO_2$ decrease earlier than end-tidal carbon dioxide tension ($P_{ET}CO_2$), but increase in $tcPCO_2$ was slower than $P_{ET}CO_2$.[16] In this case, we did not measure $P_{ET}CO_2$ because patient was not intubated. $TcPCO_2$ and $PaCO_2$ did not change significantly, when $tcPO_2$ and PaO_2 decreased. Therefore, $tcPO_2$ was the most useful to detect decrease in PaO_2.

In conclusion, $tcPO_2$ was better than SpO_2 to detect decrease in PaO_2 by pulmonary aspiration during surgery under epidural and spinal anesthesia with sedation.

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